

STIC-ILL

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QA154.VS2  
Adams

**From:** Navarro, Albert  
**Sent:** Thursday, June 12, 2003 12:37 PM  
**To:** STIC-ILL  
**Subject:** 09/518,156

Mark Navarro  
1645  
306-3225  
8A15

Dear stic

Could you please forward the following:

Vaccine 16: 768-774, 1998

Thanks

Mark

STIC-ILL

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**From:** Navarro, Albert  
**Sent:** Thursday, June 12, 2003 1:00 PM  
**To:** STIC-ILL  
**Subject:** 09/518,156

Mark Navarro  
1645  
306-3225  
8A15

Dear stic

Could you please forward the following:

Infection & Immunity November 1998, Vol. 66, No. 11, pp 5073-81

Thanks

Mark

L6 ANSWER 5 OF 9 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 3  
ACCESSION NUMBER: 1998:515640 BIOSIS  
DOCUMENT NUMBER: PREV199800515640  
TITLE: Vaccination with trypomastigote surface antigen 1-encoding  
plasmid DNA confers protection against lethal Trypanosoma  
**cruzi** infection.  
AUTHOR(S): Wizel, Benjamin; Garg, Nisha; Tarleton, Rick L.  
(1)  
CORPORATE SOURCE: (1) Univ. Georgia, Dep. Cellular Biol., 724 Biological  
Sciences Building, Athens, GA 30602 USA  
SOURCE: Infection and Immunity, (Nov., 1998) Vol. 66, No. 11, pp.  
5073-5081.  
ISSN: 0019-9567.  
DOCUMENT TYPE: Article  
LANGUAGE: English

AB DNA vaccination was evaluated with the experimental murine model of Trypanosoma **cruzi** infection as a means to induce antiparasite protective immunity, and the trypomastigote surface antigen 1 (TSA-1), a target of anti-T. **cruzi** antibody and major histocompatibility complex (MHC) class I-restricted CD8+ cytotoxic T-lymphocyte (CTL) responses, was used as the model antigen. Following the intramuscular immunization of H-2b and H-2d mice with a plasmid DNA encoding an N-terminally truncated TSA-1 lacking or containing the C-terminal nonapeptide tandem repeats, the antibody level, CTL response, and protection against challenge with T. **cruzi** were assessed. In H-2b mice, antiparasite antibodies were induced only by immunization with the DNA construct encoding TSA-1 containing the C-terminal repeats. However, both DNA constructs were efficient in eliciting long-lasting CTL responses against the protective H-2Kb-restricted TSA-1515-522 epitope. In H-2d mice, inoculation with either of the two TSA-1-expressing vectors effectively generated antiparasite antibodies and primed CTLs that lysed T. **cruzi**-infected cells in an antigen-specific, MHC class I-restricted, and CD8+-T-cell-dependent manner. When TSA-1 DNA-vaccinated animals were challenged with T. **cruzi**, 14 of 22 (64%) H-2b and 16 of 18 (89%) H-2d mice survived the infection. The ability to induce significant murine anti-T. **cruzi** protective immunity by immunization with plasmid DNA expressing TSA-1 provides the basis for the application of this technology in the design of optimal DNA multicomponent anti-T. **cruzi** vaccines which may ultimately be used for the prevention or treatment of Chagas' disease.

=>

ACCESSION NUMBER: 1997:299831 BIOSIS  
DOCUMENT NUMBER: PREV199799599034  
TITLE: The identification and molecular characterization of  
Trypanosoma **cruzi** amastigote surface protein-1 a  
member of the trans-sialidase gene super-family.  
AUTHOR(S): Santos, Maria A. M.; Garg, Nisha; **Tarleton, Rick L.**  
(1)  
CORPORATE SOURCE: (1) Dep. Cell. Biol., Univ. Georgia, Athens, GA 30602 USA  
SOURCE: Molecular and Biochemical Parasitology, (1997) Vol. 86, No.  
1, pp. 1-11.  
ISSN: 0166-6851.  
DOCUMENT TYPE: Article  
LANGUAGE: English

AB An accumulating body of evidence suggests that T. **cruzi**-infected host cells are recognized and destroyed by class I major histocompatibility complex (MHC) restricted CD8+ T-cells thus contributing to immune control of the infection. However, to date, only a few amastigote proteins which could be the target of this response have been described and gene sequence information is available only for the amastins. In order to identify amastigote proteins which could contribute to immune detection of infected host cells, a panel of monoclonal antibodies specific for amastigote proteins was produced and screened. Three mAbs (IIIC4, VIIC1 and IIID4) were identified which recognized amastigote surface proteins of 78, 26 and 53 kDa, respectively. Screening of an amastigote cDNA expression library with mAb IIIC4 resulted in the isolation of a 2.8 Kb clone, pSI2. The derived amino acid sequence indicates that the pSI2 clone encodes an amastigote surface protein belonging to the T. **cruzi** trans-sialidase super-family. Based on its preferential expression in the amastigote stage we have named this protein amastigote surface protein-1 (ASP-1). ASP-1 contains the third and fourth Asp block motifs, SxDxGxTW and the fibronectin type III-like domain, VTVxNVxLYNR, thus placing it in family II of the T. **cruzi** trans-sialidases. ASP-1 is the first trans-sialidase family member shown to be preferentially expressed in the amastigote stage of the T. **cruzi** life cycle. This expression of ASP-1 on parasites in infected cells and its apparent membrane attachment by a glycosylphosphatidylinositol (GPI)-anchor makes it a prime candidate to enter the class I MHC processing and presentation pathway.

=>

ACCESSION NUMBER: 2002468533 MEDLINE  
DOCUMENT NUMBER: 22215687 PubMed ID: 12228281  
TITLE: Genetic immunization elicits antigen-specific protective immune responses and decreases disease severity in Trypanosoma **cruzi** infection.  
AUTHOR: Garg Nisha; Tarleton Rick L  
CORPORATE SOURCE: Center for Tropical and Emerging Infectious Diseases and Department of Cellular Biology, University of Georgia, Athens, Georgia 30602, USA.  
CONTRACT NUMBER: AI33106 (NIAID)  
P01-AI44979 (NIAID)  
SOURCE: INFECTION AND IMMUNITY, (2002 Oct) 70 (10) 5547-55.  
Journal code: 0246127. ISSN: 0019-9567.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200210  
ENTRY DATE: Entered STN: 20020914  
Last Updated on STN: 20021019  
Entered Medline: 20021018

AB Immunity to Trypanosoma **cruzi** requires elicitation of humoral and cell-mediated immune responses to extracellular trypomastigotes and intracellular amastigotes. In this study, the effectiveness of the T. **cruzi** trans-sialidase family (ts) genes ASP-1, ASP-2, and TSA-1 as genetic vaccines was assessed. Immunization of mice with plasmids encoding ASP-1, ASP-2, or TSA-1 elicited poor antigen-specific cytotoxic-T-lymphocyte (CTL) activity and T. **cruzi**-specific antibody responses. Codelivery of interleukin-12 and granulocyte-macrophage colony-stimulating factor plasmids with antigen-encoding plasmids resulted in a substantial increase in CTL activity and antibody production and in increased resistance to T. **cruzi** infection. In pooled results from two to four experiments, 30 to 60% of mice immunized with antigen-encoding plasmids and 60 to 80% of mice immunized with antigen-encoding plasmids plus cytokine adjuvants survived a lethal challenge with T. **cruzi**. In comparison, 90% of control mice injected with empty plasmid DNA died during the acute phase of infection. However, the pool of three ts genes provided no greater protection than the most effective single gene (ASP-2) either with or without coadministration of cytokine plasmids. Importantly, the extent of tissue parasitism, inflammation, and associated tissue damage in skeletal muscles during the chronic phase of T. **cruzi** infection in mice immunized with antigen-encoding plasmids plus cytokine adjuvants was remarkably reduced compared to mice immunized with only cytokine adjuvants or empty plasmid DNA. These results identify new vaccine candidates and establish some of the methodologies that might be needed to develop effective vaccine-mediated control of T. **cruzi** infection. In addition, this work provides the first evidence that prophylactic genetic immunization can prevent the development of Chagas' disease.

ACCESSION NUMBER: 1999003112 MEDLINE  
DOCUMENT NUMBER: 99003112 PubMed ID: 9784506  
TITLE: Vaccination with trypomastigote surface antigen 1-encoding plasmid DNA confers protection against lethal Trypanosoma **cruzi** infection.  
AUTHOR: Wizel B; Garg N; Tarleton R L  
CORPORATE SOURCE: Department of Cellular Biology, University of Georgia, Athens, Georgia 30602, USA.  
CONTRACT NUMBER: AI33106 (NIAID)  
SOURCE: INFECTION AND IMMUNITY, (1998 Nov) 66 (11) 5073-81. Journal code: 0246127. ISSN: 0019-9567.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals; AIDS  
ENTRY MONTH: 199811  
ENTRY DATE: Entered STN: 19990106  
Last Updated on STN: 19990106  
Entered Medline: 19981123

AB DNA vaccination was evaluated with the experimental murine model of Trypanosoma **cruzi** infection as a means to induce antiparasite protective immunity, and the trypomastigote surface antigen 1 (TSA-1), a target of anti-T. **cruzi** antibody and major histocompatibility complex (MHC) class I-restricted CD8(+) cytotoxic T-lymphocyte (CTL) responses, was used as the model antigen. Following the intramuscular immunization of H-2(b) and H-2(d) mice with a plasmid DNA encoding an N-terminally truncated TSA-1 lacking or containing the C-terminal nonapeptide tandem repeats, the antibody level, CTL response, and protection against challenge with T. **cruzi** were assessed. In H-2(b) mice, antiparasite antibodies were induced only by immunization with the DNA construct encoding TSA-1 containing the C-terminal repeats. However, both DNA constructs were efficient in eliciting long-lasting CTL responses against the protective H-2Kb-restricted TSA-1515-522 epitope. In H-2(d) mice, inoculation with either of the two TSA-1-expressing vectors effectively generated antiparasite antibodies and primed CTLs that lysed T. **cruzi**-infected cells in an antigen-specific, MHC class I-restricted, and CD8(+)-T-cell-dependent manner. When TSA-1 DNA-vaccinated animals were challenged with T. **cruzi**, 14 of 22 (64%) H-2(b) and 16 of 18 (89%) H-2(d) mice survived the infection. The ability to induce significant murine anti-T. **cruzi** protective immunity by immunization with plasmid DNA expressing TSA-1 provides the basis for the application of this technology in the design of optimal DNA multicomponent anti-T. **cruzi** vaccines which may ultimately be used for the prevention or treatment of Chagas' disease.

L4 ANSWER 29 OF 31 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.  
ACCESSION NUMBER: 1998:109067 BIOSIS  
DOCUMENT NUMBER: PREV199800109067  
TITLE: Immunization with DNA containing the sequence coding for 82  
kDA protein present at the surface of Trypanosoma  
**cruzi** metacyclic trypomastigotes.  
AUTHOR(S): Boscardin, S. B.; Santori, F.; Ramirez, M. I.; Yoshida, N.;  
Franco Da Silveira, J.  
CORPORATE SOURCE: Dep. Microbiol. Imunol. Parasitol., UNIFESP, Escola  
Paulista Med., Rua Botucatu 862, CEP: 04023-062, SP Brazil  
SOURCE: Memorias do Instituto Oswaldo Cruz, (Nov., 1997) Vol. 92,  
No. SUPPL. 1, pp. 249.  
Meeting Info.: XIII Meeting of the Brazilian Society of  
Protozoology and the XXIV Annual Meeting on Basic Research  
in Chagas' Disease Caxambu, Brazil November 11-14, 1997  
Brazilian Society of Protozoology  
. ISSN: 0074-0276.  
DOCUMENT TYPE: Conference  
LANGUAGE: English

L2 ANSWER 12 OF 46 USPATFULL

ACCESSION NUMBER: 2001:102380 USPATFULL

TITLE: Cryptopain vaccines, antibodies, proteins, peptides,  
DNA and RNA for prophylaxis, treatment and diagnosis  
and for detection of cryptosporidium species

INVENTOR(S): Petersen, Carolyn, Berkeley, CA, United States

Huang, Jin-Xing, San Francisco, CA, United States

PATENT ASSIGNEE(S): The Regents of the University of California, Oakland,  
CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6254869	B1	20010703
APPLICATION INFO.:	US 1997-827171		19970327 (8)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-14233P	19960327 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Kunz, Gary L.	
ASSISTANT EXAMINER:	Gucker, Stephen	
LEGAL REPRESENTATIVE:	Verny, Hana	
NUMBER OF CLAIMS:	12	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	10 Drawing Figure(s); 9 Drawing Page(s)	
LINE COUNT:	1424	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD A **DNA** or RNA **vaccine** for prevention and treatment of  
infections caused by **protozoan** Cryptosporidium species  
(Cryptosporidium) in humans and other mammals was developed by utilizing  
newly identified and isolated DNA (SEQ ID NOs: 1-3) and amino acid  
sequences of the Cryptosporidium pathogen designated cryptopain.

=>



L2 ANSWER 20 OF 46 USPATFULL

ACCESSION NUMBER: 2000:7383 USPATFULL

TITLE: Vaccines, antibodies, proteins, glycoproteins, DNAs and RNAs for prophylaxis and treatment of Cryptosporidium parvum infections

INVENTOR(S): Petersen, Carolyn, Berkeley, CA, United States  
Leech, James, Daly City, CA, United States  
Nelson, Richard C., San Francisco, CA, United States  
Gut, Jiri, Novato, CA, United States

PATENT ASSIGNEE(S): The Regents of the University of California, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6015882		20000118
APPLICATION INFO.:	US 1996-700651		19960814 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1995-415751, filed on 3 Apr 1995, now patented, Pat. No. US 5643772 which is a continuation of Ser. No. US 1993-71880, filed on 1 Jun 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-891301, filed on 29 May 1992, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Housel, James C.		
ASSISTANT EXAMINER:	Portner, Ginny Allen		
LEGAL REPRESENTATIVE:	Verny, Hana		
NUMBER OF CLAIMS:	17		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	14 Drawing Figure(s); 10 Drawing Page(s)		
LINE COUNT:	2686		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD The anti-Cryptosporidium vaccine of the invention contains a Cryptosporidium antigen identified by the invention, modified in such a way that it is incapable of producing the Cryptosporidium symptoms but is capable of eliciting the production of specific protective antibodies against the disease when introduced in the body. A **DNA** or RNA **vaccine** for prevention and treatment of infections caused by **protozoan** Cryptosporidium species (Cryptosporidium) in humans and other mammals was developed by utilizing newly identified and isolated DNA (SEQ ID NOs: 1-4) and amino acid (SEQ ID NOs: 5 and 6) sequences of the Cryptosporidium pathogen designated GP900.

L22 ANSWER 10 OF 31 USPATFULL

ACCESSION NUMBER: 2002:116050 USPATFULL  
TITLE: Auxiliary gene and protein of methicillin resistant  
bacteria and antagonists thereof  
INVENTOR(S): Tomasz, Alexander, New York, NY, United States  
De Lencastre, Herminia, New York, NY, United States  
PATENT ASSIGNEE(S): The Rockefeller University, New York, NY, United States  
(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6391614	B1	20020521
APPLICATION INFO.:	US 1999-419163		19991015 (9)
RELATED APPLN. INFO.:	Division of Ser. No. US 1996-679635, filed on 10 Jul 1996, now patented, Pat. No. US 5985643		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-19315P	19960607 (60)
	US 1995-1042P	19950710 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Carlson, Karen Cochrane	
LEGAL REPRESENTATIVE:	Klauber & Jackson	
NUMBER OF CLAIMS:	9	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	12 Drawing Figure(s); 12 Drawing Page(s)	
LINE COUNT:	2101	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM The invention is further directed to a **DNA** molecule comprising a nucleic acid sequence which encodes a **protein** associated with **antibiotic** resistance in a *S. aureus* bacterium, which nucleic acid sequence is preferably located in the SmaI-I fragment of the chromosome of the *S. aureus* bacterium. In particular, the **DNA** molecule comprises in gene, a **mutation** which results in a blockade of cell wall synthesis at or close to the branch point in hexose metabolism involved in the synthesis of cell wall components. The compositional change of peptidoglycan in a mutant of the invention is the complete disappearance of the unsubstituted disaccharide pentapeptide monomer. In a specific embodiment, the mutant corresponds to RUSA315. In a still further embodiment, the gene has a nucleotide sequence as depicted in FIG. 6 (SEQ ID NO: 1).